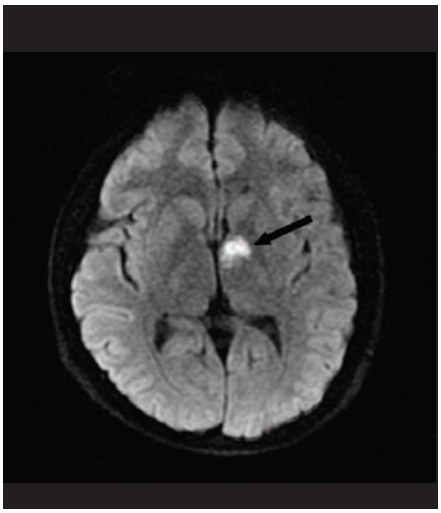

[CASE REPORT]

Visual Hallucinations Following a Left-sided Unilateral Tuberothalamic Artery Infarction

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KEY WORDS: Cerebrovascular accident, stroke, brain infarction, encephalopathy, visual hallucinations, anomia, dysnomia, left tuberothalamic artery, absence of motor or sensory deficits, diagnostic accuracy, central nervous system pathology

ABSTRACT

A 20-year-old man presented with realistic visual hallucinations and no motor or sensory neurological findings. The absence of motor or sensory deficits on physical examination made for a diagnostic challenge, but an altered mental status with dysnomic word-finding difficulty was the clue to the existence of an encephalopathy that resulted in evaluation for structural pathology. Brain imaging revealed an infarction in the territory of the left tuberothalamic artery. A head magnetic resonance imaging scan identified the neuropathology that led to immediately starting treatment for stroke.

INTRODUCTION

The thalamus is a "relay station" for sensory information from many sensory areas projecting to the cortex.[1] Therefore, injury to the thalamus may result in multimodal sensory abnormalities. The tuberothalamic artery supplies the intralaminar nucleus, ventral internal medullary lamina, and mammillothalamic tract in the paramedian thalamus.[2] Isolated tuberothalamic infarctions are rare, but follow small vessel occlusion.[2] Persistence of cognitive and mental status changes after tuberothalamic artery infarction are documented.[3]

The clinical vignette presents a case of an acute encephalopathy with bizarre visual hallucinations and language difficulties, in the absence of motor or sensory neurological deficits on physical examination. These findings were initially mistaken for a psychiatric disorder, which could have delayed diagnostic and therapeutic intervention.

CLINICAL VIGNETTE

A 20-year-old, right-handed man with mental status change and abnormal behavior of three days duration was hospitalized on a psychiatry service. Severe headache

was reported in the weeks prior to presentation. He evidenced amnesia for recent events. The patient complained of frightening visual hallucinations and was agitated. He described overtly seeing "dangerous hands and snakes" of a realistic and threatening nature. His past medical and family history revealed no neurological or psychiatric disorders and no diabetes, hypertension, or substance abuse.

On mental status examination, the patient was awake, attentive, and cooperative. Behavior was appropriate, but he expressed a feeling of derealization and depersonalization. Speech was fluent and comprehension was normal; yet, he otherwise evidenced significant word finding difficulty. Impaired ability at naming was confirmed by using the Boston Naming Test.[4,5] The physical and neurological examination revealed difficulty with language, but without other motor, sensory, oculomotor, or visual deficits, and no spatial neglect. Routine laboratory assessments were all within normal ranges (i.e., serum chemistry, lipid profile, a hemogram, and a urinalysis). Results of an electrocardiogram and cerebrospinal fluid studies were unremarkable. A head scan by magnetic resonance imaging (MRI) revealed an acute unilateral infarction of the left tuberothalamic artery territory (Figures 1 and 2). Once the infarct was diagnosed, the patient became a neurology case and all appropriate stroke therapies and rehabilitation services were instituted. See Table 1 for signs of tuberothalamic artery infarction.

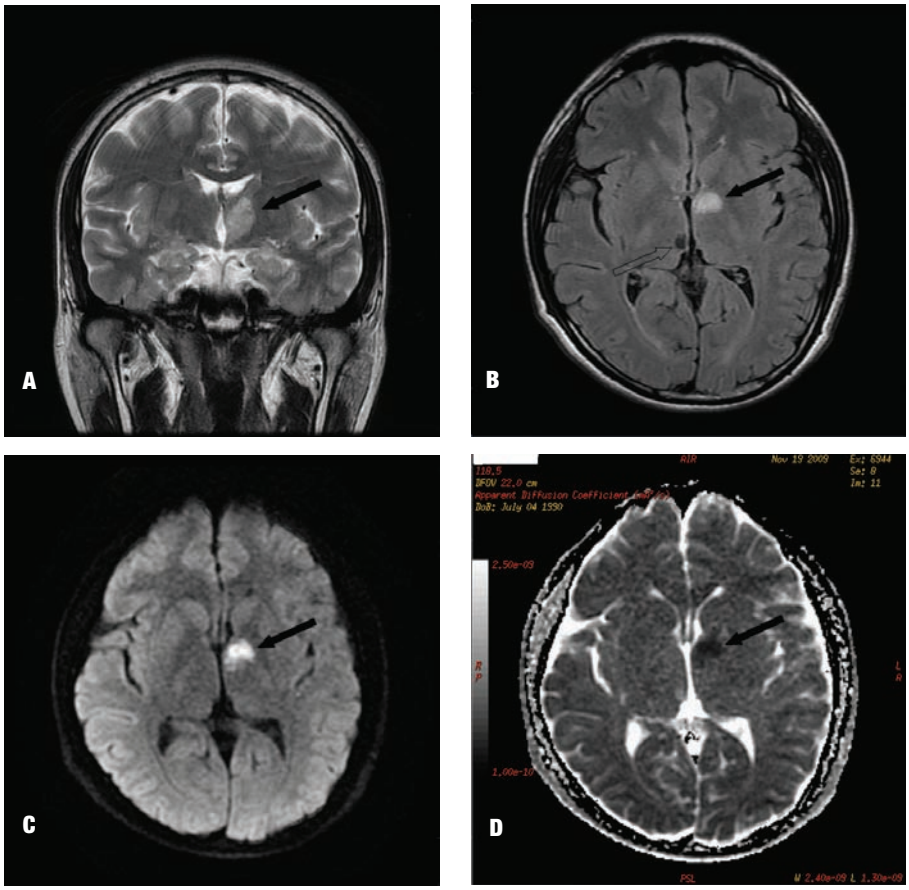

FIGURE 1. Coronal fast-spin echo T2-weighted images (1.5 Tesla, TR 4000ms, TE 125ms, slice thickness 5mm, 320 3 224 matrix, NEX 2) and an axial FLAIR (1.5 Tesla, TR 4000ms, TE 125ms, slice thickness 5mm, 320 3 224 matrix, NEX 2, fluid attenuation inversion recovery time 5 2000 msec) revealed a high signal intensity lesion in the anterior portion of the left thalamus (*black arrows*) territory as illustrated in A and B, respectively. The shape and location of this lesion matches the tuberothalamic artery territory. The diffusion-weighted image (*b* 5 1000) and apparent diffusion coefficient image evidenced a high signal intensity (*black arrow*) and low signal intensity (*black arrow*) of the lesion, respectively, suggesting that this lesion has the character of a diffusion restriction, or that this finding is an infarction, as seen in C and D, respectively. This patient also had a lacuna (*open arrow*) in the right thalamus as noted in B.

DISCUSSION

An altered mental status can result from a cerebrovascular accident.[6] The most common psychiatric disturbances resulting from a stroke include cognitive and/or speech impairments with dementia and/or delirium, depression, mania, and anxiety; involuntary emotional expression disorder (i.e., pathological laughter or crying) can also be observed.[7] Left hemisphere strokes frequently cause aphasia or dysnomias, as observed in our case, while right hemisphere strokes are associated with anosognosia, inattention, impaired spatial reasoning, and neglect syndromes.[7] Tuberothalamic artery infarction can produce impairments of arousal, orientation, learning or memory, personality, and executive functioning.[2,3]

A unilateral tuberothalamic syndrome can be differentiated neurologically and neuropsychologically from other thalamic lesions, because in contrast to posterolateral and paramedian thalamic lesions, patients with ischemia in the tuberothalamic artery region exhibit no marked disorders of consciousness or attention, nor vertical gaze paresis or oculomotor deficits.[8]

Peduncular hallucinosis is a rare neurological condition presenting with repeated visual hallucinations resulting from a lesion in the midbrain, pons, and/or thalamic regions, without ocular pathology.[9]

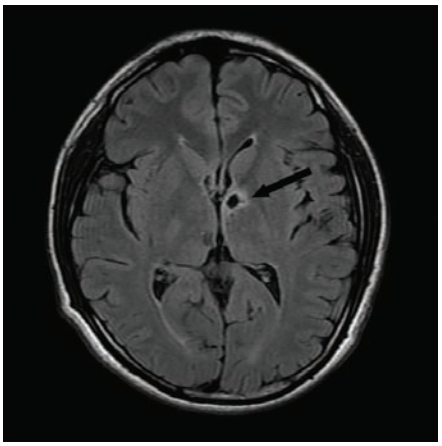

FIGURE 2. The axial FLAIR image (1.5 Tesla, TR 4000ms, TE 125ms, slice thickness 5mm, 320 3 224 matrix, NEX 2, fluid attenuation inversion recovery, IR 5 2000 msec) revealed a fluid-filled cavity with a high signal intensity rim, illustrating the evolution of the infarction (*black arrow*) to a lacuna after three weeks.

TABLE 1. Signs of tuberothalamic artery infarction[2,3,8]

• fluctuation in arousal and orientation	
• impaired memory, learning, and comprehension	
• language dysfunction	
• acalculia	
Right-sided features	Left-sided features
• apraxia	• aphasia, dysphasia
• dyspraxia	• paraphasia
• left-sided neglect	• dysarthria • anomia, dysnomia • monotone voice • perseveration

Vascular, infectious, or neoplastic diseases are the usual etiologies. These hallucinations frequently are bright images that occur nocturnally, typically, but not always, during sleep, and not consistently perceived as being part of reality. If or when the neuropathology is corrected, perceptions tend to normalize.

Aberrant perceptions associated with psychiatric illnesses are more commonly interpreted as real life events and are generally an observation during wakefulness. For optimal treatment, it is critical to differentiate between hallucinosis due to medical, neurological, and/or psychiatric causations. Visual hallucinations are most often associated with encephalopathies of a toxic, metabolic, or neurologic origin. This also applies to tactile or olfactory hallucinations, while auditory hallucinations are typically based on psychopathology.

Our described patient's stroke resulted in a picture compatible with peduncular hallucinosis, and cerebrovascular pathology often can induce such a clinical picture.[5] However, less characteristic of peduncular hallucinosis was its daytime, wakefulness occurrence, and that the patient perceived the visual phenomena as a bothersome reality. In fact, he reported frightening visual images that scared him. There was no evidence of these being an illusion, sensory phenomenon, or hallucinations of a hypnogogic or hypnopompic type.

This case presented with visual hallucinations as a major symptom in the absence of motor or sensory deficits, a somewhat uncommonly encountered feature. Such a presentation can be misdiagnosed as a psychiatric disorder, thus delaying stroke treatment. Our patient exhibited a change in mental status, and absence of abnormal motor or sensory signs made determination of the correct diagnosis more difficult. The history, speech disturbance, and memory impairment characterized an encephalopathy that was the clue to precipitate an investigation for brain pathology and discovery of the tuberothalamic artery infarct. The patient had a neurological disease despite a presentation that was initially thought to have been a mental illness.

The tuberothalamic artery originates from the middle third of the posterior communicating artery, but may be absent in 30 to 40 percent of the population.[3] When present, it supplies the reticular nucleus, the mammillothalamic tract, part of the ventral lateral nucleus, the dorsomedial nucleus, and the lateral aspect of the thalamic pole.[3,10] MRI studies suggest that hallucinations can be caused by a lesion in the thalamic reticular nucleus.[11] A unilateral thalamic lesion could cause hallucinations, and the intralaminar and dorsomedial nuclei pathology might explain this symptom pattern.[12]

Our patient presented with difficulties in retrieving object names and evidenced an altered mental status. His impaired verbal memory could be ascribed to pathology in the mammillothalamic tract and the dorsomedial nucleus. Although the contribution of the thalamus to naming problems remains poorly understood, it is thought to play an important role in activating and coordinating the left temporal lobe in semantic processing at the left frontal language area, which is crucial to verbal output.[13–15] Additionally, his amnesia is explainable by involvement of Papez circuit, which includes the hippocampus, fornix, mamillary body, mammillothalamic tract, anterior thalamic nuclei, and the cingulate gyrus. This circuit plays a role in memory function.[16]

Mental illness symptoms may mask neurological conditions and vice versa. Although the incidence of a brain infarct is rare in young adults, physicians should always consider brain pathology in evaluating patients with new onset psychiatric issues, particularly when signs of an encephalopathy are present.[17] Prompt diagnosis and intervention in cases of a stroke are always important; since young, brain-injured individuals are more likely to recover comprehension than older ones, early recognition for them is especially crucial.[18,19]

Physicians sometimes tend to dismiss neurological diagnoses from their differential considerations when the physical examination reveals few signs for motor or sensory deficits. Finding only subtle abnormalities in the neurological system can lead to diagnostic confusion, as seen in our vignette; however, the phasic

dysfunction was the feature together with amnesia and visual hallucinosis that led to obtaining the studies identifying the brain infarct.[17]

Our patient evidenced a dysnomia with an inability to recognize the meaning of words in a form compatible with our case clinically and neuropathologically. Language disturbances in the left hemisphere are characterized by various degrees of naming difficulties with potentially decreased verbal output, fluent paraphasic speech or impaired fluency, and poor comprehension. Semantic and phonemic paraphasic errors occur, with occasional neologisms and perseveration. Reading can be preserved, although the comprehension may be poor. In contrast, repetition is preserved. These characteristics of language impairment are consistent with thalamic aphasias, although some features may vary and the specific contribution of each thalamic nucleus to the impaired linguistic element is less clear.[3]

CONCLUSION

An altered mental status with dysnomia and visual hallucinations of a menacing type, but without abnormal motor or sensory findings on physical examination in a young man, led ultimately to the diagnosis of a left unilateral tuberothalamic infarction. This lesion demonstrates the role of a specific brain structure in the expression of visual hallucinations. Doctors must fully screen patients for cerebral pathology in such encephalopathic presentations. To enhance diagnostic precision and to provide timely treatment, a brain imaging study should be performed immediately to rule out brain disease when structural pathology is in the differential.

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